Journal of Zoo and Wildlife Medicine 45(3): 632–634, 2014

LONG-TERM ASSESSMENT OF GLUCOSURIA IN CAPTIVE OKAPI (*OKAPIA JOHNSTONI*) AFTER A DIETARY CHANGE

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Abstract: Glucosuria in okapis (*Okapia johnstoni*) was first documented in 1980, yet the etiology remains unclear. In August 2006, an attempt to lower glucosuria in captive okapi by diet modification (omitting all fruit and adding unmolassed beet pulp) was started at the Antwerp Zoo. To study the possible relationship between glucosuria and diet, stress, and/or pregnancy, four okapis were monitored over a period of 4.5 yr. One animal, born in 2006, became glucosuric near the age of three. Three okapis were adults at the start of the study and had been glucosuric for more than 5 yr. The glucose/creatinine urinary ratio values of these four glucosuric animals did not change considerably over time despite dietary changes. Stress did not appear to influence glucosuria in these okapi. Urinary ratio decreased during the second half of pregnancy in two females. In conclusion, the diet change did not reduce glucosuria, but pregnancy appeared to lower urinary glucose in okapis.

Key words: Glucosuria, diet, okapi, *Okapia johnstoni*, pregnancy.

BRIEF COMMUNICATION

Since the initial observation of glucosuria in okapis (*Okapia johnstoni*) in 1980,[5] attempts have been made to explain the etiology. Excretion of glucose in the urine of mammals is not normally observed but can be seen in cases of diabetes mellitus, renal tubule disorders, or renal disease.[4] One major etiology considered in okapi is diabetes mellitus; however, glucosuric okapis had normal serum levels of insulin, glucose, and fructosamine.[4] Fanconi syndrome with dysfunction of the proximal renal tubules is another possible etiology;[2] however, in okapis this was ruled out because animals were clinically normal and urine specific gravity and blood levels of phosphorous and potassium were within normal ranges.[15] Although glomerulonephritis is not uncommon in adult okapis,[6] most glucosuric okapis are clinically healthy, which may suggest a dietary influence.

No glucosuria was noted in managed okapi at the Okapi Breeding and Research Station located near the village of Epulu in the Democratic Republic of Congo. Those animals received a natural diet of 23.0 kg leaves of diverse species, daily per animal on an as-fed basis.[4] In most zoos, the availability of leaves may be limited. Okapi managed at the Zoo of Antwerp were fed a diet (original diet) containing 2.0% vegetables, 5.5% fruit, 13.5% pellets, and 79.0% lucerne hay on a dry matter basis (DMB). This diet likely had elevated concentrations of sugars and starches compared with a diet containing predominantly leaves. An initial dietary trial using lower amounts of sugars and starch appeared to lower urinary glucose values in one female okapi.[14] Therefore, starting in August 2006 and implementing results of research on digestive physiology and feeding of okapi,[8,9] the diet of okapi at the Antwerp Zoo was modified (new diet) to include 0.3% vegetables, 0.0% fruit, 10.9% pellets, 14.2% leaves, 16.8% unmolassed beet pulp, and 57.8% lucerne hay (DMB).

Four glucosuric okapis at the Zoo of Antwerp were followed between August 2006 and February 2011 (4.5 yr total). At the start of the study, the male (M) was 6 yr old, female 1 (F1) was 9 yr old (gave birth on 17 September 2008), female 2 (F2) was 15 yr old (was not pregnant during the observation period), and female 3 (F3) was 2 mo old (gave birth on 04 July 2011, after the end of the observation period). All adults (M, F1, and F2) were fed the original diet for more than 5 yr, while one female (F3) received the new diet from early preweaning because of her young age. Free-catch, midstream urine samples were collected opportunistically. Urine glucose and creatinine were quantitatively measured by dry biochemistry (Kodak–Johnson & Johnson, 2340 Beerse, Belgium), and glucose/creatinine urinary ratio (UR) was calculated to account for changes in urine concentration.[3] Animals were considered glucosuric if UR was greater than one.[4] Fecal pregnanediol and 11,17-dioxoandrostanes were

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Table 1. Median (minimum–maximum) values of the glucose/creatinine urinary ratio (UR) in *N* samples of one male (M) and 3 female (F1, F2, and F3) okapi (*Okapia johnstoni*) on a new diet for a period of 4.5 yr.

Okapi	*N*	UR new diet[a]	*N*	UR fecal cortisol and pregnanediol[b]
M	18	12.15 (2.73–20.88)	7	11.67 (2.73–16.37)
F1	44	15.72 (1.49–32.22)	7	19.46 (1.49–29.29)
F2	61	5.08 (0.66–9.64)	7	6.53 (3.61–8.86)
F3	51	1.00 (0.33–7.50)	11	3.04 (1.00–4.56)

[a] UR values after the diet change.
[b] UR values after the diet change on days of fecal cortisol and pregnanediol analysis.

analyzed at the University of Veterinary Medicine (Vienna, Austria) as previously described with enzyme immunoassays for measuring progestogens and cortisol, respectively.[12,13]

Median, minimum, and maximum UR values did not change considerably for any of the animals (Table 1). The UR in the male (M) increased steadily, which was not observed in the females (Fig. 1). Coincidentally, the male's UR decreased during the two pregnancies of F1 and F3 monitored during the study. A possible explanation could be that the male was mating several other females during these periods; however, there are no reports in literature linking mating activity with glucosuria in any animal species. F3 became glucosuric near the age of three, but urinary glucose showed a general decreasing trend during its pregnancy, which continued beyond the observation period (Fig. 1). During the first 19 mo on the new diet, UR decreased in F1 and F2;[15] however, it began to rise again thereafter in F2. UR of F1 showed a general decreasing trend during its entire pregnancy until delivery, when UR increased (Fig. 1). Evidently, the hypothesis of a direct link between diet and glucosuria could not be sustained.

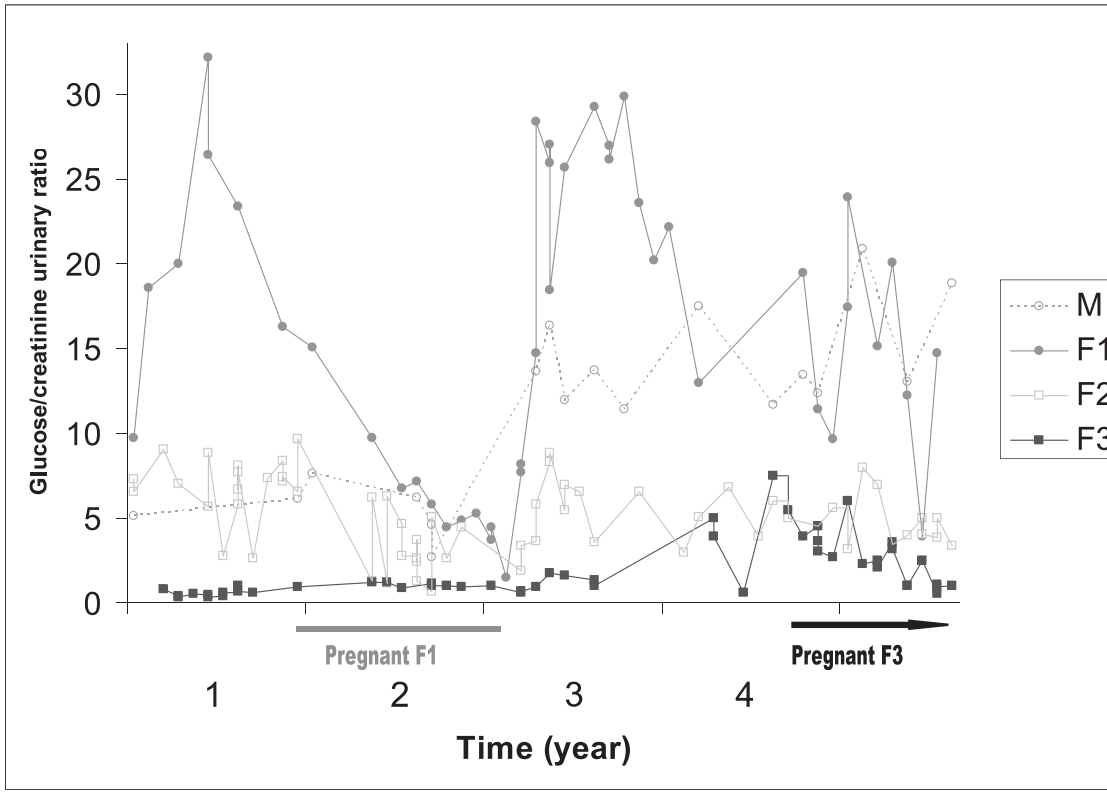

Figure 1. The glucose/creatinine urinary ratio of one male (M) and three female (F1, F2, and F3) okapi (*Okapia johnstoni*) during 4.5 yr on the new diet with pregnancies indicated.

The possibility that stress, caused by daily intraspecific contact in these naturally solitary animals[1] through the potential insulin-opposing effect of glucocorticoids,[10] was excluded because fecal cortisol metabolites (11.17-dioxoandrostanes) did not substantially change the UR. Diagnostics to rule out diabetes or glomerulonephritis were not performed.

Interestingly, the UR of females F1 and F3 showed a general decreasing trend during their pregnancy until delivery, while fecal progestogens (pregnanediol) increased. According to Harding,[7] glucose is the major fetal energy substrate in sheep, and the ovine placenta takes up 60% of the glucose from the uterine circulation during late gestation. This may explain the decrease in UR observed in both pregnant okapis.

Benign (or familial) renal glucosuria in humans is a genetic disorder and presents without hyperglycemia or proximal tubular dysfunction. It is caused by mutations in the gene encoding for the sodium-coupled glucose transporter, SGLT2.[11] This condition shows an autosomal recessive inheritance,[11] and although there is practically no inbreeding in the okapi population,[4,15] it may contribute to the explanation of glucosuria.

In conclusion, the new diet with decreased amounts of sugars and starch did not reduce glucosuria. Similarly, stress did not seem to have an impact; however, gestation did seem to decrease glucosuria. Other potential etiologies, such as benign/familial renal disease or the influence of pregnancy on glucose metabolism, could explain observed glucosuria in okapis; however, these possibilities need further research.

Acknowledgments: The authors wish to thank Dr. M. Clauss (Vetsuisse Faculty, University of Zurich, Switzerland), Dr. J. Nijboer (Okapi nutrition advisor), and Dr. F. Schwarzenberger (University of Veterinary Medicine, Vienna, Austria) for their advice; Dr. A. Moresco (Denver Zoo, United States) for editing the scientific language and grammar; the Flemish Government for structural support to the Centre for Research and Conservation; the Okapi International Studbook (Dr. K. Leus and S. Hofman); and the okapi keepers at the zoo for their efforts at collecting samples. The authors are also grateful to the reviewers and the associate editor for their helpful and very constructive comments on the original version of this manuscript.

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Received for publication 8 June 2012